

Laparoscopic Treatment of Superior Mesenteric Artery Syndrome

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ABSTRACT

Background: Superior mesenteric artery syndrome is caused by compression of the third portion of the duodenum by the superior mesenteric artery. Many disease states predispose one to this condition.

Methods: We present a case report of a young female patient who presented with gastro-duodenal obstruction from superior mesenteric artery syndrome and subsequently underwent surgical treatment with minimally invasive techniques. Pathophysiology of SMA syndrome is reviewed.

Results: The cause of superior mesenteric artery syndrome is variable but always results in duodenal obstruction. Surgery is one treatment option that is effective and can be performed laparoscopically.

Conclusion: Laparoscopic duodenojejunostomy is an acceptable method of treating superior mesenteric artery syndrome.

Key Words: Laparoscopic duodenojejunostomy, Superior mesenteric artery syndrome.

INTRODUCTION

Superior mesenteric artery (SMA) syndrome is a condition of duodenal obstruction secondary to external compression of the third portion of the duodenum. Patients typically present with bilious vomiting, early satiety, and postprandial epigastric pain and fullness. Results of studies published in the 1980s documented only 400 cases. It is a difficult disease to diagnose because of its rarity and its insidious nature. SMA syndrome is often a diagnosis of exclusion. It should be considered in patients who have chronic abdominal complaints and risk factors for developing this condition. These predisposing factors will be reviewed. Treatment options range from medical to surgical interventions. We present a case report of SMA syndrome and a surgical treatment option utilizing minimally invasive techniques.

CASE REPORT

A 21-year-old Japanese female presented to the emergency room with complaints of abdominal pain for 1 day. The onset of sharp pain was sudden, increasing in severity, and relieved briefly by an unknown Japanese medication. The pain started superior to the umbilicus and then radiated to the entire abdomen. It increased while the patient was lying supine. Her last bowel movement was approximately 12 hours prior to admission and was normal in caliber and consistency. She denied passing flatus since then. The patient felt nauseated and vomited once after her last meal, approximately 24 hours prior to admission. She had been afebrile.

The patient related a 2-year history of bulimia and a 6-year history of anorexia. She described vomiting 1 to 2 times per day. The patient had no other past medical or surgical history, took an unknown Japanese medication for her abdominal pain, and had no allergies or family history significant to her presentation. She did report drinking approximately 2 ounces of alcohol 5 to 6 times per day. She was living alone in the United States with all of her family still in Japan. She was G0P0 and was sexually active, with her last menstrual period 2 months prior to admission.

On examination, the patient was hemodynamically stable

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with vital signs as follows: blood pressure 129/92, heart rate 88, respirations 18, temperature 99.7°F, and oxygen saturation 98%. She weighed 90 lbs and stood 5'1", with a body mass index of 17 kg/m², making her 15 lbs under ideal body weight. A cardiopulmonary examination was normal. Bowel sounds were present; but the abdomen was distended and diffusely tender, but with no rebound or guarding. Pelvic examination results were normal.

Metabolic panel, hematologic profile, coagulation studies, liver function tests, and B-hCG results were all normal. An upright abdominal film showed a large stomach displacing the abdominal contents, with small amounts of air throughout the colon (**Figure 1**). A computed tomography scan of the abdomen demonstrated dilation of the stomach and proximal duodenum with a transition point at the third portion of the duodenum (**Figure 2**). No obvious intraluminal, mural, or extrinsic mass was present. A nasogastric tube was placed, and approximately 1800 mL of slightly bilious fluid was aspirated over 4 hours. The patient was treated non-surgically with continued nasogastric decompression and started on total parenteral nutrition. An upper gastrointestinal series was performed that showed a narrowing of the third segment of the duodenum in the region of the superior mesenteric

artery (**Figure 3**). A subsequent angiogram showed a patent SMA traversing across the duodenum, correlating to the site of obstruction, with moderate compression of the celiac trunk by the arcuate ligament.

After managing the patient conservatively for 5 days with no improvement in gastric emptying, the decision was made to proceed with surgical treatment. With the patient



Figure 1. Upright abdominal x-ray demonstrating caudal displacement of the transverse colon by the fluid filled and enlarged stomach.



Figure 2. Computed tomography scan showing dilated stomach and proximal duodenum with a transition point at the third portion of the duodenum.



Figure 3. Upper gastrointestinal series showing narrowing in the third portion of the duodenum in the region of the superior mesenteric artery.

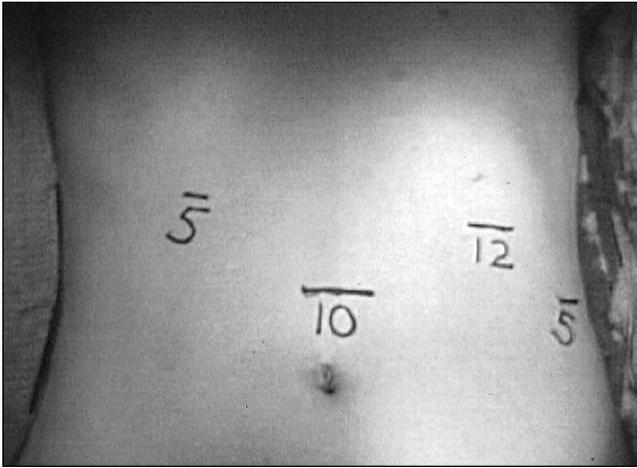


Figure 4. Placement of 12-mm supraumbilical port, 12-mm left lower quadrant port, 5-mm right subcostal port, and 5-mm left lateral port.

under general anesthesia, a laparoscopic duodenojejunostomy was performed. The abdomen was entered under direct vision via a left subcostal incision in the midclavicular line using a 12-mm Optiview trocar. After insufflation of the abdomen, a 10-mm, 30-degree laparoscope was used to maximize visualization. Three additional trocars were placed: a 12-mm supraumbilical port, a 12-mm left lower quadrant port and a 5-mm right subcostal port (**Figure 4**). The stomach and proximal duodenum were noted to be significantly dilated. After retraction of the transverse colon superiorly, the ligament of Treitz was identified. The adjacent superior mesenteric artery was noted to be prominent, and lying across the distal duodenum, which was collapsed (**Figure 5**).

A duodenojejunostomy was performed using jejunum approximately 25 cm distal to the ligament of Treitz. A side-to-side anastomosis was created with a 45-mm, 2.5 endoscopic linear stapler, and 3-0 monofilament sutures for closure of the enterotomy. A methylene blue test showed no leak, and a nasogastric tube was left in the stomach. Operative time was 120 minutes with minimal blood loss. An upper gastrointestinal series performed 3 days after surgery demonstrated emptying of gastric contrast through the anastomosis into the jejunum. The patient was subsequently advanced to clear liquids and progressed to a regular diet 2 days later. The patient returned to Japan to be with her family and receive psychiatric care after discharge from the hospital.



Figure 5. Superior mesenteric artery lying across the collapsed distal duodenum.

DISCUSSION

Superior mesenteric artery syndrome has been described since the 1800s. It has been referred to as Wilkie's syndrome, the cast syndrome and arteriomesenteric duodenal compression.¹ It is a rare condition that affects less than .4% of the population. Although a consensus exists that superior mesenteric artery syndrome consists of duodenal compression by the superior mesenteric artery, there are many theories about the cause of the condition including the following:

- decrease in the SMA-aortic angle (normally 45 degrees) to less than 25 degrees¹
- postoperative rearrangement of the anatomy around the third part of the duodenum that compromises the superior mesenteric-duodenal relationship²
- low origin of the SMA or a high insertion of the duodenum at the ligament of Treitz
- lumbar lordosis
- loss of muscle tone in the duodenum
- persistent supine position
- peritoneal adhesions from duodenal malrotation causing direct compression or indirect compression via the SMA.³
- loss of mesenteric and retroperitoneal fat, which normally cushions the superior mesenteric artery anteriorly from the duodenum

Many conditions can predispose one to a decrease in aortomesenteric angle. These can be divided into 3 cate-

gories: (1) diseases associated with rapid loss of retroperitoneal and mesenteric fat, such as burns and neoplasms, (2) trauma that causes changes in innervation of the abdominal wall or spine, and (3) dietary disorders such as anorexia nervosa or malabsorptive conditions. Several medical conditions that may lead to SMA syndrome include lymphoma, tuberculosis, pancreatic malignancy, diabetes mellitus, scleroderma, systemic lupus erythematosus, and amyloidosis.⁴

SMA syndrome is diagnosed based on 3 criteria: a dilated duodenum, an aortomesenteric angle less than 25 degrees, and compression of the third part of the duodenum by the SMA.⁵ The diagnosis may be first suggested after an abdominal plain film shows a massively dilated stomach. Abdominal computed tomography scan with lateral reconstructions can be used to make the diagnosis in many cases because the site of duodenal compression and the angle of the SMA can all be examined. Endoscopy is helpful to rule out mechanical obstruction. If the diagnosis is still in question, an upper gastrointestinal series can show compression of the third portion of the duodenum, to-and-fro movement of barium proximal to the obstruction, delays of gastroduodenal transit of 4 to 6 hours, and relief of obstruction with the patient in a knee-chest or left lateral decubitus position. Arteriography can be used as a final modality to measure the aortomesenteric angle more precisely.^{1,2,6}

Medical therapy should be attempted prior to surgery. Nasogastric decompression and total parenteral nutrition are suggested for up to 7 days in some reports.⁵ Small meals are then attempted with the patient using a knee-chest positioning to help relieve the obstruction by widening the aortomesenteric angle. Enteral feedings past the ligament of Treitz can also assist in restoring mesenteric fat.⁶ Surgery is indicated if medical therapy has failed, if there is a long history of weight loss, and if peptic ulcer disease exists concurrently. Surgical treatments include gastrojejunostomy, duodenojejunostomy, and division of the ligament of Treitz with mobilization of the duodenum.¹ Minimally invasive techniques can be applied to

traditional surgical treatment to decrease wound complications, postoperative pain, and recovery time. These patients are typically thin and have anatomy conducive to laparoscopy. Laparoscopic treatment with a duodenojejunostomy has been successfully described and may become the standard of care for treatment of SMA syndrome.⁵

CONCLUSION

SMA syndrome is a rare but serious surgical condition that should be considered whenever chronic bilious vomiting exists with any of the predisposing conditions described above. The diagnostic workup includes abdominal computed tomography scan, esophagogastroduodenoscopy, upper gastrointestinal series, and angiography, if necessary. When medical therapy fails, laparoscopic duodenojejunostomy can be successfully used to bypass the third portion of the duodenum with minimal morbidity.

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